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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2	JAN 08	CHEMLIST enhanced with New Zealand Inventory of Chemicals
NEWS 3	JAN 16	CA/CAplus Company Name Thesaurus enhanced and reloaded
NEWS 4	JAN 16	IPC version 2007.01 thesaurus available on STN
NEWS 5	JAN 16	WPIDS/WPINDEX/WPIX enhanced with IPC 8 reclassification data
NEWS 6	JAN 22	CA/CAplus updated with revised CAS roles
NEWS 7	JAN 22	CA/CAplus enhanced with patent applications from India
NEWS 8	JAN 29	PHAR reloaded with new search and display fields
NEWS 9	JAN 29	CAS Registry Number crossover limit increased to 300,000 in multiple databases
NEWS 10	FEB 15	PATDPASPC enhanced with Drug Approval numbers
NEWS 11	FEB 15	RUSSIAPAT enhanced with pre-1994 records
NEWS 12	FEB 23	KOREAPAT enhanced with IPC 8 features and functionality
NEWS 13	FEB 26	MEDLINE reloaded with enhancements
NEWS 14	FEB 26	EMBASE enhanced with Clinical Trial Number field
NEWS 15	FEB 26	TOXCENTER enhanced with reloaded MEDLINE
NEWS 16	FEB 26	IFICDB/IFIPAT/IFIUDB reloaded with enhancements
NEWS 17	FEB 26	CAS Registry Number crossover limit increased from 10,000 to 300,000 in multiple databases
NEWS 18	MAR 15	WPIDS/WPIX enhanced with new FRAGHITSTR display format
NEWS 19	MAR 16	CASREACT coverage extended
NEWS 20	MAR 20	MARPAT now updated daily
NEWS 21	MAR 22	LWPI reloaded
NEWS 22	MAR 30	RDISCLOSURE reloaded with enhancements
NEWS 23	MAR 30	INPADOCDB will replace INPADOC on STN
NEWS 24	APR 02	JICST-EPLUS removed from database clusters and STN

NEWS EXPRESS	NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS LOGIN	Welcome Banner and News Items
NEWS IPC8	For general information regarding STN implementation of IPC 8

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FILE COVERS 1907 - 30 Apr 2007  VOL 146 ISS 19
FILE LAST UPDATED: 29 Apr 2007  (20070429/ED)

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=> s inducible nitric oxide synthase
         64793 INDUCIBLE
        186014 NITRIC
             3 NITRICS
        186017 NITRIC
                  (NITRIC OR NITRICS)
       1752238 OXIDE
        356029 OXIDES
       1852864 OXIDE
                  (OXIDE OR OXIDES)
        103797 SYNTHASE
          5996 SYNTHASES
        104884 SYNTHASE
                  (SYNTHASE OR SYNTHASES)
L1        8934 INDUCIBLE NITRIC OXIDE SYNTHASE
                  (INDUCIBLE(W)NITRIC(W)OXIDE(W)SYNTHASE)

=> s l1 and inflammator?
        177669 INFLAMMATOR?
L2        2688 L1 AND INFLAMMATOR?

=> s l2 and diseas?
       1070477 DISEAS?
L3        1470 L2 AND DISEAS?

=> s l3 and central nervous system
        401077 CENTRAL
            29 CENTRALS
        401100 CENTRAL
                  (CENTRAL OR CENTRALS)
        217702 NERVOUS
       2425212 SYSTEM
       1328273 SYSTEMS
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       3287324 SYSTEM
                (SYSTEM OR SYSTEMS)
         81625 CENTRAL NERVOUS SYSTEM
                (CENTRAL(W)NERVOUS(W)SYSTEM)
L4          90 L3 AND CENTRAL NERVOUS SYSTEM

=> s l4 and py<2003
      22885160 PY<2003
L5          40 L4 AND PY<2003

=> d ibib abs hitstr 1-10

L5   ANSWER 1 OF 40  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:        2002:899394  CAPLUS
DOCUMENT NUMBER:         138:378830
TITLE:                   Inhibitors of poly(ADP-ribose) polymerase-1 suppress
                         transcriptional activation in lymphocytes and
                         ameliorate autoimmune encephalomyelitis in rats
AUTHOR(S):               Chiarugi, Alberto
CORPORATE SOURCE:        Department of Preclinical and Clinical Pharmacology,
                         University of Florence, Florence, Italy
SOURCE:                  British Journal of Pharmacology (2002),
                         137(6), 761-770
                         CODEN: BJPCBM; ISSN: 0007-1188
PUBLISHER:               Nature Publishing Group
DOCUMENT TYPE:           Journal
LANGUAGE:                English
AB   In the presence of genotoxic stress poly(ADP-ribose) polymerase-1 (PARP-1)
     leads to NAD+ and ATP depletion, participating in the pathogenesis of
     several disorders including inflammation. Accordingly, chemical inhibitors
     of PARP-1 are efficacious anti-inflammatories, albeit the
     underlying mol. mechanisms are still under debate. This study
     investigated the effect of the PARP-1 inhibitors 6(5H)-phenanthridinone
     and benzamide as well as that of benzoic acid, an inactive analog of
     benzamide, on development of exptl. allergic encephalomyelitis (EAE) in
     rats. Both 6(5H)-phenanthridinone and benzamide attenuated development of
     EAE, reducing clin. score, neuroimmune infiltration and expression of
     inflammatory mediators such as inducible nitric
     oxide synthase, interleukin-1β and -2,
     cyclooxygenase-2, tumor necrosis factor-α and interferon-γ in
     the spinal cord of myelin-immunized rats. Importantly, no evidence of
     NAD+ and ATP depletion as well as poly(ADP-ribose) formation was detected
     in the spinal cord. By contrast, a robust formation of poly(ADP-ribose)
     occurred in B- and T-cell areas in lymph nodes of myelin-immunized rats
     and was suppressed by the treatment with 6(5H)-phenanthridinone and
     benzamide. In cultures of activated rat lymphocytes, 6(5H)-
     phenanthridinone and benzamide reduced the DNA-binding activity of
     NF-κB and AP-1 and transcription of pro- inflammatory
     cytokines such as interleukin-2, interferon-γ and tumor necrosis
     factor-α. Notably, benzoic acid did not reproduce the in vivo and
     in vitro effects of its parent compound These findings indicate that PARP-1
     promotes transcriptional activation in lymphocytes and inhibitors of its
     enzymic activity are useful for the treatment of autoimmune disorders of
     the central nervous system.
REFERENCE COUNT:         56    THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5   ANSWER 2 OF 40  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:        2002:741590  CAPLUS
DOCUMENT NUMBER:         138:34588
TITLE:                   Ethanol-Induced Modulation of Inducible
                         Nitric Oxide Synthase
                         Activity in Human A172 Astrocytoma Cells
```

AUTHOR(S): Davis, Randall L.; Dertien, Janet; Syapin, Peter J.
CORPORATE SOURCE: Alcohol and Brain Research Laboratory, Department of Pharmacology, Texas Tech University Health Sciences Center, Lubbock, TX, USA
SOURCE: Alcoholism: Clinical and Experimental Research (2002), 26(9), 1404-1411
CODEN: ACRSDM; ISSN: 0145-6008
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Background: Glial cells are critical in the functioning of the central nervous system (CNS), including responsiveness to injury and immunocompetence. The immune and inflammatory response involves the inducible form of nitric oxide synthase (iNOS), and subsequent NO production Previously, the authors have demonstrated that ethanol inhibits cytokine-induced iNOS expression and activity in rat glial cells. Evidence of ethanol-induced effects on iNOS in human glial cells is nonexistent. Herein, the conditions necessary for significant iNOS induction in human A172 astrocytoma cells have been characterized, and subsequently, the effects of ethanol on iNOS expression have been investigated. Methods: A172 cells were analyzed immunohistochem. for the astrocyte markers, glial fibrillary acidic protein (GFAP) and S-100β. The ability of A172 cells to express iNOS was assessed by stimulating cells with interferon-γ (IFNγ), tumor necrosis factor-α (TNFα), interleukin-1β (IL-1β), bacterial lipopolysaccharide (LPS), L-arginine, and tetrahydrobiopterin (BH4) in various combinations. Following stimulation, iNOS induction was monitored via measurement of nitrite production and in vitro iNOS enzyme activity. Time-course (6-24 h) studies assessed the effects of ethanol (50-200 mM) on iNOS induction. Results: Immunohistochem. anal. confirmed that A172 cells were phenotypically, astrocytic. Induction of nitrite production by a cytomix [IFNγ (100 ng/mL) +TNFα (30 ng/mL) + IL-1β (5 ng/mL)] was differentially enhanced by exposure to supplemental factors including LPS, L-arginine, and BH4. Nitrite production was greatest over the initial 24 h of stimulation with iNOS enzyme activity peaking at 12 h. Acute (6-24 h) exposure of activated cells to 50 mM ethanol enhanced iNOS activity recovered from the cytosol, whereas 200 mM ethanol decreased it. Ethanol had no direct effect on the catalytic activity of the enzyme. Conclusions: The present study is the first published report of ethanol-induced modulation of iNOS expression in human glial cells. The data suggest that ethanol is influencing iNOS enzyme levels most profoundly. Altered astrocyte function may be a point of ethanol-induced perturbation in CNS immune function. These findings should lend insight into the role of ethanol on human CNS immunity and brain injury.

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:719058 CAPLUS
DOCUMENT NUMBER: 138:3199
TITLE: M-CSF deficiency leads to reduced metallothioneins I and II expression and increased tissue damage in the brain stem after 6-aminonicotinamide treatment
AUTHOR(S): Penkowa, Milena; Poulsen, Christian Bjorn; Carrasco, Javier; Hidalgo, Juan
CORPORATE SOURCE: Department of Medical Anatomy, The Panum Institute, University of Copenhagen, Copenhagen, DK-2200, Den.
SOURCE: Experimental Neurology (2002), 176(2), 308-321
CODEN: EXNEAC; ISSN: 0014-4886
PUBLISHER: Elsevier Science
DOCUMENT TYPE: Journal

LANGUAGE: English

AB 6-Aminonicotinamide (6-AN) is a niacin antagonist, which leads to degeneration of gray-matter astrocytes followed by a vigorous inflammatory response. Macrophage colony stimulating factor (M-CSF) is important during inflammation, and to further clarify the roles for M-CSF in neurodegeneration and brain cell death, the authors have examined the effect of 6-AN on osteopetrotic mice with genetic M-CSF deficiency (op/op mice). The 6-AN-induced degeneration of graymatter areas was comparable in control and op/op mice, but the nos. of reactive astrocytes, macrophages, and lymphocytes in the damaged areas were significantly decreased in op/op mice relative to controls. The levels of oxidative stress (as determined by using immunoreactivity for inducible nitric oxide synthase, nitrotyrosine, and malondialdehyde) and apoptotic cell death (as determined by using TUNEL and immunoreactivity for caspases and cytochrome c) were significantly increased in 6-AN-injected op/op mice relative to controls. From a number of antioxidant factors assayed, only metallothioneins I and II (MT-I+II) were decreased in op/op mice in comparison to controls. Thus, the present results indicate that M-CSF is an important growth factor for coping with 6-AN-induced central nervous system damage and suggest that MT-I+II are likely to have a significant role.

REFERENCE COUNT: 93 THERE ARE 93 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 4 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:694982 CAPLUS

DOCUMENT NUMBER: 138:265333

TITLE: Discovery of new chemical classes of synthetic ligands that suppress neuroinflammatory responses

AUTHOR(S): Watterson, D. Martin; Haiech, Jacques; Van Eldik, Linda J.

CORPORATE SOURCE: Drug Discovery Program and Departments of Molecular Pharmacology and Biological Chemistry, Northwestern University Medical School, Chicago, IL, 60611-3008, USA

SOURCE: Journal of Molecular Neuroscience (2002), 19(1/2), 89-93
CODEN: JMNEES; ISSN: 0895-8696

PUBLISHER: Humana Press Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The authors used a chemical genomics approach that includes follow up in parallel syntheses to discover a new class of compds. that selectively suppress glial activation. While the mechanism of action remains to be determined, available data and the exptl. approach for discovery indicate that the mechanism includes inhibition of gene regulating protein kinases. Specifically, the increased production of IL-1β and iNOS in response to various activating stimuli, including Aβ1-42, is suppressed while the production of potentially beneficial responses, such as ApoE production, is not inhibited. The increased production of COX-2 and p38 MAPK activation are also not altered, demonstrating the novel nature of potential therapeutic targets compared to currently available drugs. The chemical scaffold is 3-aminopyridazine (3-AP). This is an attractive scaffold because of its potential for diversification by established, facile chemistries and the prior use of a 3-AP scaffold in other central nervous system targeted therapeutics. Therefore, the potential bioavailability of 3-AP derivs. and the demonstrated cellular selectivity demand that future research address the potential efficacy of selective 3-AP derivs. in animal models of disease.

REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 5 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:594628 CAPLUS
DOCUMENT NUMBER: 137:150265
TITLE: Substituted aryl compounds as novel cyclooxygenase-2 selective inhibitors, compositions and methods of use
INVENTOR(S): Khanapure, Subhash P.; Garvey, David S.; Earl, Richard A.; Ezawa, Maiko; Fang, Xinqin; Gaston, Ricky D.
PATENT ASSIGNEE(S): Nitromed, Inc., USA
SOURCE: PCT Int. Appl., 132 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002060378	A2	20020808	WO 2001-US48823	20011221 <--
WO 2002060378	A3	20031231		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2432642	A1	20020808	CA 2001-2432642	20011221 <--
AU 2002249812	A1	20020812	AU 2002-249812	20011221 <--
US 2002119977	A1	20020829	US 2001-24046	20011221 <--
US 6706724	B2	20040316		
EP 1406609	A2	20040414	EP 2001-998052	20011221
EP 1406609	B1	20060906		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
JP 2005502587	T	20050127	JP 2002-560574	20011221
AT 338544	T	20060915	AT 2001-998052	20011221
US 2004116431	A1	20040617	US 2003-730979	20031210
US 6825185	B2	20041130		
US 2005059665	A1	20050317	US 2004-969079	20041021

PRIORITY APPLN. INFO.:

US 2000-256932P	P	20001221
US 2001-24046	A3	20011221
WO 2001-US48823	W	20011221
US 2003-730979	A1	20031210

OTHER SOURCE(S): MARPAT 137:150265

AB Substituted aryl compds. that are cyclooxygenase 2 (COX-2) selective inhibitors and compns. comprising at least one COX-2 selective inhibitor, and, optionally, at least one compound that donates, transfers or releases nitric oxide, stimulates endogenous synthesis of nitric oxide, elevates endogenous levels of endothelium-derived relaxing factor or is a substrate for nitric oxide synthase, and/or, optionally, at least one therapeutic agent are described. A therapeutic agent is selected from steroids, nonsteroidal anti-inflammatory compds. (NSAID), 5-lipoxygenase (5-LO) inhibitors, leukotriene B4 (LTB4) receptor antagonists, leukotriene A4 (LTA4) hydrolase inhibitors, 5-HT agonists, 3-hydroxy-3-methylglutaryl CoA (HMG-CoA) inhibitors, H2 antagonists, antineoplastic agents, antiplatelet agents, thrombin inhibitors, thromboxane inhibitors, decongestants, diuretics, sedating or non-sedating antihistaminics, inducible nitric oxide synthase inhibitors, opioids, analgesics, Helicobacter pylori inhibitors, proton pump inhibitors, and isoprostane inhibitors. The invention also provides novel kits comprising at least one COX-2 selective inhibitor, and,

optionally, at least one nitric oxide donor, and/or, optionally, at least one therapeutic agent. The cyclooxygenase-2 selective inhibitors of the invention can be optionally nitrosated and/or nitrosylated. The invention also provides methods for treating inflammation, pain and fever; for treating and/or improving the gastrointestinal properties of COX-2 selective inhibitors; for facilitating wound healing; for treating and/or preventing renal toxicity or other toxicities; for treating and/or preventing other disorders resulting from elevated levels of cyclooxygenase-2; and for improving the cardiovascular profile of COX-2 selective inhibitors.

L5 ANSWER 6 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:458947 CAPLUS
DOCUMENT NUMBER: 137:123894
TITLE: Intracerebroventricular but not intravenous interleukin-1β induces widespread vascular-mediated leukocyte infiltration and immune signal mRNA expression followed by brain-wide glial activation
AUTHOR(S): Proescholdt, M. G.; Chakravarty, S.; Foster, J. A.; Foti, S. B.; Briley, E. M.; Herkenham, M.
CORPORATE SOURCE: Section on Functional Neuroanatomy, National Institute of Mental Health, Bethesda, MD, 20892-4070, USA
SOURCE: Neuroscience (Oxford, United Kingdom) (2002), 112(3), 731-749
CODEN: NRSCDN; ISSN: 0306-4522
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Interleukin-1β (IL-1β) is a pro- inflammatory cytokine that appears in brain and cerebrospinal fluid following peripheral immune challenges and central infections or injury. The authors examined the consequences of i.c.v. infusion of IL-1β on mRNA expression of several immune markers and on recruitment of peripheral leukocytes. Awake rats were infused with IL-1β (100 ng/rat) into the lateral ventricle, and 0.5, 2, 4, 8, 12, or 24 h later, animals were killed and their fresh-frozen brains processed for in situ hybridization and immunohistochem. Widespread vascular expression of inhibitory factor κBα (IκBα, marker of nuclear factor κBα transcriptional activity) and inducible cyclooxygenase (COX-2) mRNAs at 0.5-2 h was credited to movement of IL-1β along ventricular, subarachnoid, and perivascular pathways to target endothelia that express type 1 IL-1 receptor mRNA. Induction of monocyte chemoattractant protein-1 mRNA and intercellular adhesion mol.-1 (ICAM-1) immunostaining on endothelia began at 0.5-2 h. Leukocytes (neutrophils and monocytes, recognized by morphol. and CD45 and ED1 immunostaining) appeared in meninges and blood vessels at 2-4 h and diffusely penetrated the parenchyma at 8-24 h. The leukocytes strongly expressed IL-1β and inducible nitric oxide synthase mRNAs. Beginning at 4-12 h, astrocytes (glial acidic fibrillary protein mRNA and protein and c-fos mRNA) and microglia (ionized calcium-binding adaptor mol. 1 mRNA and protein) showed widespread activation. Other rats received i.v. IL-1β (6 μg/kg). Their brains showed induction of IκBα and COX-2 mRNAs in the vasculature at 2 h but none of the other sequelae. In summary, the data indicate that IL-1β in the cerebrospinal fluid reaches its target receptors on the endothelia via perivascular volume transmission, up-regulates ICAM-1, and triggers a targeted leukocyte emigration and widespread glial activation stimulated perhaps by pro-inflammatory mols. expressed by leukocytes. The dramatic difference between i.c.v. and i.v. routes of administration underscores the potency of IL-1β within the brain to dynamically affect the cellular trafficking component of 'immune privilege'.
REFERENCE COUNT: 68 THERE ARE 68 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 7 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:382961 CAPLUS
DOCUMENT NUMBER: 137:62013
TITLE: Role of mitogen-activated protein kinases in inducible nitric oxide synthase and TNFα expression in human fetal astrocytes
AUTHOR(S): Hua, Liwei L.; Zhao, Meng-Liang; Cosenza, Melissa; Kim, Mee-Ohk; Huang, Huan; Tanowitz, Herbert B.; Brosnan, Celia F.; Lee, Sunhee C.
CORPORATE SOURCE: Department of Pathology, Albert Einstein College of Medicine, Bronx, NY, 10461, USA
SOURCE: Journal of Neuroimmunology (2002), 126(1-2), 180-189
CODEN: JNRIDW; ISSN: 0165-5728
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Astrocytes are important sources of proinflammatory mediators such as iNOS and TNFα in the diseased central nervous system. In previous studies, we showed that the cytokine IL-1 plays a critical role in the activation of human astrocytes to express TNFα and the inducible form of nitric oxide synthase (iNOS). In the present study, we have addressed the role of the MAP-kinase pathway in the signaling events leading to the induction of these genes. Treatment with SB203580, a specific inhibitor of p38 mitogen-activated protein kinases (MAPK), potently inhibited IL-1-mediated induction of iNOS and TNFα in cultures of human fetal astrocytes. In contrast, PD98059, an upstream inhibitor of the extracellular regulated kinase (ERK)1/2 pathway, had little or no effect. Interestingly, SB203580 reduced the mRNA expression for iNOS, TNFα, and IL-6, indicating inhibition prior to translation. Transfection of astrocytes with a dominant-neg. Jun-NH2-terminal kinase (JNK) construct also reduced iNOS expression. Western blot anal. showed phosphorylated p38 and JNK in IL-1-activated astrocytes, and phosphorylated ERK in both resting and activated cells. Electrophoretic mobility shift assay (EMSA) showed that IL-1 induced NF-κB and AP-1 DNA complex formation in astrocytes, and that SB203580 inhibited AP-1 complex formation. These results demonstrate the differential roles played by the three MAP kinases in human astrocyte inflammatory gene activation and point to a crucial function of p38 and JNK MAP kinases in IL-1-mediated astrocyte activation.
REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 8 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:366268 CAPLUS
DOCUMENT NUMBER: 137:199764
TITLE: Identification of new therapeutic targets for prevention of CNS inflammation
AUTHOR(S): Owens, Trevor
CORPORATE SOURCE: Neuroimmunology Unit, Montreal Neurological Institute, Montreal, QC, H3A 2B4, Can.
SOURCE: Expert Opinion on Therapeutic Targets (2002), 6(2), 203-215
CODEN: EOTTAO; ISSN: 1472-8222
PUBLISHER: Ashley Publications Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Multiple sclerosis (MS) is a disease of complex pathologies, which involves infiltration by CD4+ and CD8+ T cells of and response within the central nervous system.

Expression in the CNS of cytokines, reactive nitrogen species and costimulator mols. have all been described in MS. Notably, the cytokines IFN-γ and TNF are strongly expressed. Microglial cells in the CNS express costimulator mols. and it is assumed that they play a role in directing or inducing the T cell response. Transgenic expts. have tested the effects of overexpression of these mols. in mice and have shown that TNF has multiple effects in the CNS. These range from pro-inflammatory effects of soluble TNF signalling through one of its receptors TNF-RI, to protective/regenerative effects of membrane-associated TNF signalling through the other receptor, TNF-RII. Although IFN-γ induces nitric oxide production via the enzyme inducible nitric oxide synthase, which is immunosuppressive, IFN-γ is predominantly pro- inflammatory . In CNS disease in mice that involves CD8+ T cells, IFN-γ blockade is protective. Finally, microglial expression of the costimulator ligand B7.2 induces demyelinating pathol. Animal expts. therefore point to IFN-γ and costimulatory microglia as logical targets of therapy for MS. IFN-γ represents a more accessible target and should therefore be pursued at the earliest opportunity.

REFERENCE COUNT: 89 THERE ARE 89 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 9 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:294227 CAPLUS
DOCUMENT NUMBER: 136:315023
TITLE: Polydithiocarbamate-containing nontargeting macromolecules for therapeutic and diagnostic applications
INVENTOR(S): Lai, Ching-san
PATENT ASSIGNEE(S): Medinox, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 18 pp., Cont.-in-part of U.S. Ser. No. 899,087, abandoned.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 2002045573	A1	20020418	US 1999-409645		19991001 <--
US 6649591	B2	20031118			
CN 1230178	A	19990929	CN 1997-197797		19970828 <--
KR 2000035992	A	20000626	KR 1999-701945		19990309 <--
PRIORITY APPLN. INFO.:			US 1996-25867P	P	19960910
			US 1997-899087	B2	19970723

OTHER SOURCE(S): MARPAT 136:315023

AB A new class of drugs for treatment of such indications as cerebral stroke and other ischemia/reperfusion injury is disclosed. Dithiocarbamates are linked to the surface of a non-immunogenic, nontargeting macromol. other than an antibody (e.g., albumin) either by using crosslinking reagents or by nonspecific binding to produce polydithiocarbamate-macromol.-containing compns., which represent a new class of drugs for treatment of such indications as cerebral stroke and other ischemia/reperfusion injury. Combinational therapeutic methods have been developed for the in vivo inactivation or inhibition of formation (either directly or indirectly) of species which induce the expression of inducible nitric oxide synthase, as well as reducing nitric oxide levels produced as a result of NO synthase expression. Magnetic resonance imaging methods have been developed for the measurement of cerebral and cardiac blood flow and infarct volume in ischemic stroke or heart attack situations. Such methods employ iron-containing complexes of a composition comprising a dithiocarbamate and a nonimmunogenic, nontargeting macromol.

other than an antibody as contrast agents. The crosslinking expts. were performed by the treatment of bovine serum albumin (BSA) with N-hydroxysulfosuccinimidyl-4-azido salicylic acid in DMSO at pH 7.0. The reaction mixture was incubated at ambient temperature for 10-60 min. Upon the addition of N-methyl-D-glucamine dithiocarbamate (MGD), the solution was irradiated at 365 nm for 1-5 min. After irradiation, the solution was applied onto a G-25 pre-packed column. The MGD-BSA containing fractions were collected and rechromatographed once. The stoichiometry of MGD bound to the BSA mol. can be estimated by measuring the absorbance at 215 nm (for MGD) and 280 nm (for BSA).

L5 ANSWER 10 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:151387 CAPLUS
DOCUMENT NUMBER: 137:134928
TITLE: Water-soluble chitosan inhibits the production of pro-inflammatory cytokine in human astrocytoma cells activated by amyloid β peptide and interleukin-1β
AUTHOR(S): Kim, Mi-Sun; Sung, Man-Joon; Seo, Sang-Bong; Yoo, Su-Jin; Lim, Woon-Ki; Kim, Hyung-Min
CORPORATE SOURCE: Department of Oriental Pharmacy, Wonkwang University, College of Pharmacy and Korea Institute of Oriental Pharmacy, Chonbuk, Iksan, 570-749, S. Korea
SOURCE: Neuroscience Letters (2002), 321(1-2), 105-109
CODEN: NELED5; ISSN: 0304-3940
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A chronic inflammatory response associated with β-amyloid (Aβ) and interleukin-1β (IL-1β) is responsible for the pathol. of Alzheimer's disease (AD). Astrocytes are predominant neuroglial cells of the central nervous system and are actively involved in cytokine-mediated events in AD. To investigate the biol. effect of water-soluble chitosan (WSC), we examined cytotoxicity, production of pro-inflammatory cytokines and inducible nitric-oxide synthase (iNOS) on human astrocytoma cell line CCF-STTG1 stimulated with IL-1β and Aβ fragment 25-35 (Aβ[25-35]). In 3-[4,5-dimethylthiazol-2-yl]-2,5-diphenyltetrazolium bromide colorimetric assay, WSC by itself had no effect on cell viability on human astrocytoma cells. The effects of WSC on tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6) were evaluated with ELISA and Western blotting. The production of TNF-α and IL-6 was induced by IL-1β and Aβ[25-35] and synergistically amplified by the co-stimulation of IL-1β and Aβ[25-35]. The secretion and expression of pro- inflammatory cytokines, TNF-α and IL-6, was significantly inhibited by pretreatment with WSC in human astrocytoma cells. The expression of iNOS was induced by IL-1β and Aβ[25-35] and was partially inhibited by treatment with WSC. We demonstrate the regulatory effects of WSC in human astrocytes for the first time and suggest the anti-inflammatory effect of WSC may reduce and delay AD pathol. events.
REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> FIL STNGUIDE

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	50.28	50.49

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION

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CA SUBSCRIBER PRICE                                          -7.80       -7.80

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AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Apr 27, 2007 (20070427/UP).

=> d his

     (FILE 'HOME' ENTERED AT 10:23:11 ON 30 APR 2007)

     FILE 'CAPLUS' ENTERED AT 10:23:34 ON 30 APR 2007
L1          8934 S INDUCIBLE NITRIC OXIDE SYNTHASE
L2          2688 S L1 AND INFLAMMATOR?
L3          1470 S L2 AND DISEAS?
L4            90 S L3 AND CENTRAL NERVOUS SYSTEM
L5            40 S L4 AND PY<2003

     FILE 'STNGUIDE' ENTERED AT 10:26:02 ON 30 APR 2007

=> log y
COST IN U.S. DOLLARS                                  SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.24      50.73

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)            SINCE FILE      TOTAL
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CA SUBSCRIBER PRICE                                       0.00      -7.80

STN INTERNATIONAL LOGOFF AT 10:28:41 ON 30 APR 2007
```

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSPTANXR1625

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2	JAN 08	CHEMLIST enhanced with New Zealand Inventory of Chemicals
NEWS 3	JAN 16	CA/CAplus Company Name Thesaurus enhanced and reloaded
NEWS 4	JAN 16	IPC version 2007.01 thesaurus available on STN
NEWS 5	JAN 16	WPIDS/WPINDEX/WPIX enhanced with IPC 8 reclassification data
NEWS 6	JAN 22	CA/CAplus updated with revised CAS roles
NEWS 7	JAN 22	CA/CAplus enhanced with patent applications from India
NEWS 8	JAN 29	PHAR reloaded with new search and display fields
NEWS 9	JAN 29	CAS Registry Number crossover limit increased to 300,000 in multiple databases
NEWS 10	FEB 15	PATDPASPC enhanced with Drug Approval numbers
NEWS 11	FEB 15	RUSSIAPAT enhanced with pre-1994 records
NEWS 12	FEB 23	KOREAPAT enhanced with IPC 8 features and functionality
NEWS 13	FEB 26	MEDLINE reloaded with enhancements
NEWS 14	FEB 26	EMBASE enhanced with Clinical Trial Number field
NEWS 15	FEB 26	TOXCENTER enhanced with reloaded MEDLINE
NEWS 16	FEB 26	IFICDB/IFIPAT/IFIUDB reloaded with enhancements
NEWS 17	FEB 26	CAS Registry Number crossover limit increased from 10,000 to 300,000 in multiple databases
NEWS 18	MAR 15	WPIDS/WPIX enhanced with new FRAGHITSTR display format
NEWS 19	MAR 16	CASREACT coverage extended
NEWS 20	MAR 20	MARPAT now updated daily
NEWS 21	MAR 22	LWPI reloaded
NEWS 22	MAR 30	RDISCLOSURE reloaded with enhancements
NEWS 23	MAR 30	INPADOCDB will replace INPADOC on STN
NEWS 24	APR 02	JICST-EPLUS removed from database clusters and STN

NEWS EXPRESS	NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS LOGIN	Welcome Banner and News Items
NEWS IPC8	For general information regarding STN implementation of IPC 8

Enter NEWS followed by the item number or name to see news on that specific topic.

* * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 09:59:36 ON 30 APR 2007

=> file reg
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'REGISTRY' ENTERED AT 09:59:47 ON 30 APR 2007
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PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

Property values tagged with IC are from the ZIC/VINITI data file
provided by InfoChem.

STRUCTURE FILE UPDATES: 27 APR 2007 HIGHEST RN 933069-51-3
DICTIONARY FILE UPDATES: 27 APR 2007 HIGHEST RN 933069-51-3

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH December 2, 2006

Please note that search-term pricing does apply when
conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document. For information
on property searching in REGISTRY, refer to:

http://www.cas.org/support/stngen/stndoc/properties.html

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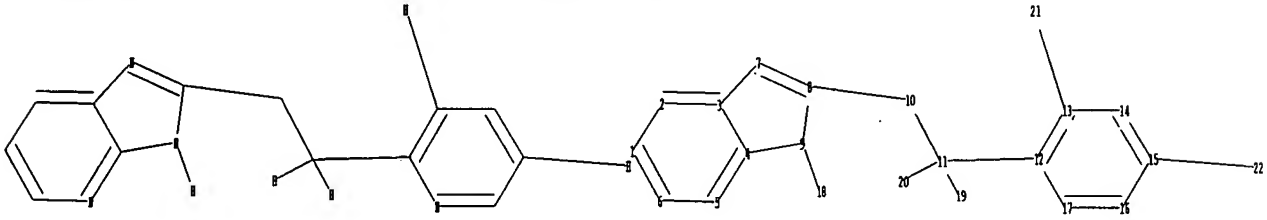

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10 11 18 19 20 21 22
ring nodes :
1 2 3 4 5 6 7 8 9 12 13 14 15 16 17
chain bonds :
8-10 9-18 10-11 11-12 11-19 11-20 13-21 15-22
ring bonds :
1-2 1-6 2-3 3-4 3-7 4-5 4-9 5-6 7-8 8-9 12-13 12-17 13-14 14-15 15-16
16-17
exact/norm bonds :
3-7 4-9 7-8 8-9
exact bonds :
8-10 9-18 10-11 11-12 11-19 11-20 13-21 15-22
normalized bonds :
1-2 1-6 2-3 3-4 4-5 5-6 12-13 12-17 13-14 14-15 15-16 16-17
isolated ring systems :
containing 1 : 12 :

G1:C,H

Match level :
1:Atom 2:Atom 3:Atom 4:Atom 5:Atom 6:Atom 7:Atom 8:Atom 9:Atom 10:CLASS
11:CLASS 12:Atom 13:Atom 14:Atom 15:Atom 16:Atom 17:Atom 18:CLASS 19:CLASS
20:CLASS 21:CLASS 22:CLASS

L1 STRUCTURE UPLOADED

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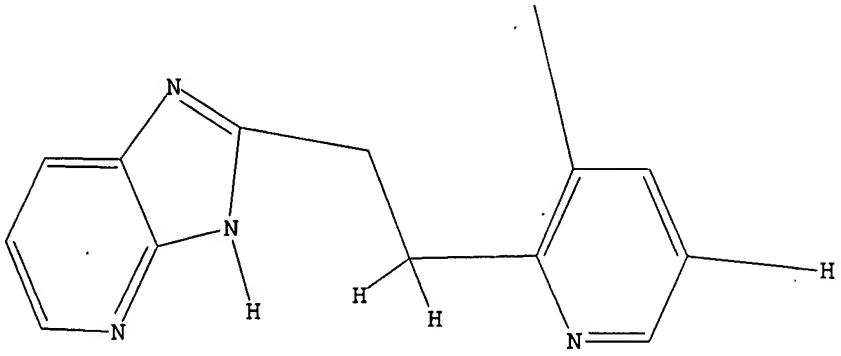
L1 HAS NO ANSWERS
L1 STR



G1 C,H

Structure attributes must be viewed using STN Express query preparation.

=> s l1
SAMPLE SEARCH INITIATED 10:00:14 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED - 38 TO ITERATE

100.0% PROCESSED 38 ITERATIONS 5 ANSWERS
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS: ONLINE **COMPLETE**
BATCH **COMPLETE**
PROJECTED ITERATIONS: 391 TO 1129
PROJECTED ANSWERS: 5 TO 234

L2 5 SEA SSS SAM L1

=> s l1 full
FULL SEARCH INITIATED 10:00:19 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED - 769 TO ITERATE

100.0% PROCESSED 769 ITERATIONS 161 ANSWERS
SEARCH TIME: 00.00.01

L3 161 SEA SSS FUL L1

=> file caplus
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION

FULL ESTIMATED COST 172.10 172.31

FILE 'CAPLUS' ENTERED AT 10:00:23 ON 30 APR 2007
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FILE COVERS 1907 - 30 Apr 2007 VOL 146 ISS 19
FILE LAST UPDATED: 29 Apr 2007 (20070429/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply.
They are available for your review at:

http://www.cas.org/infopolicy.html

=> s l3 full
L4 9 L3

=> s l9 and py<2003
L9 NOT FOUND
The L-number entered could not be found. To see the definition of L-numbers, enter DISPLAY HISTORY at an arrow prompt (=>).

=> s l4 and py<2003
22885160 PY<2003
L5 1 L4 AND PY<2003

=> d ibib abs hitstr tot

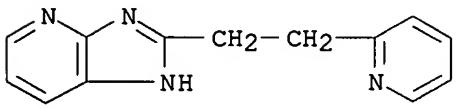
L5 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1999:284034 CAPLUS
DOCUMENT NUMBER: 131:82668
TITLE: Synthesis and antiproliferative activity of some N-sulfonated-2-substituted benzimidazoles and imidazo[4,5-b]pyridines
AUTHOR(S): Garuti, Laura; Varoli, Lucilla; Cermelli, Claudio; Baggio, Giosue; Lupo, Lucia; Malagoli, Monica; Castelli, Mario
CORPORATE SOURCE: Department of Pharmaceutical Science, University of Bologna, Bologna, I-40126, Italy
SOURCE: Anti-Cancer Drug Design (1998), 13(8), 969-980
CODEN: ACDDEA; ISSN: 0266-9536
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Some N-sulfonated-2-substituted benzimidazoles and imidazo[4,5-b]-pyridines were synthesized and tested in vitro for antiviral and antiproliferative activity. None of the compds. had antiviral properties. However, 3 of them inhibited the proliferation of leukemia and lymphoma cell lines at micromolar concns. The maximum potency of antiproliferative activity is correlated with the presence of an ethylenic spacer between

the 2 heterocycles.

IT 229468-73-9P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(synthesis and antiproliferative activity of N-sulfonated-2-substituted benzimidazoles and imidazo[4,5-b]pyridines)

RN 229468-73-9 CAPLUS

CN 1H-Imidazo[4,5-b]pyridine, 2-[2-(2-pyridinyl)ethyl]- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> FIL STNGUIDE

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	7.75	180.06

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-0.78	-0.78

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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Apr 27, 2007 (20070427/UP).

=> file reg

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.12	180.18

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-0.78

FILE 'REGISTRY' ENTERED AT 10:02:32 ON 30 APR 2007
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STRUCTURE FILE UPDATES: 27 APR 2007 HIGHEST RN 933069-51-3
DICTIONARY FILE UPDATES: 27 APR 2007 HIGHEST RN 933069-51-3

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TSCA INFORMATION NOW CURRENT THROUGH December 2, 2006

Please note that search-term pricing does apply when

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REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

http://www.cas.org/support/stngen/stndoc/properties.html

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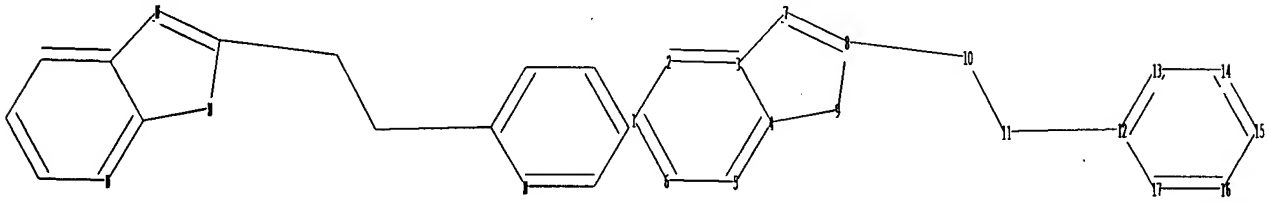


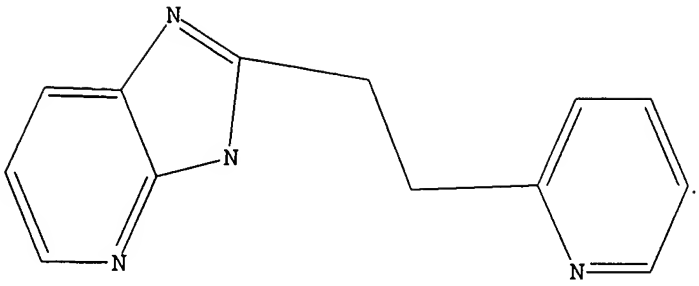
chain nodes :
10 11
ring nodes :
1 2 3 4 5 6 7 8 9 12 13 14 15 16 17
chain bonds :
8-10 10-11 11-12
ring bonds :
1-2 1-6 2-3 3-4 3-7 4-5 4-9 5-6 7-8 8-9 12-13 12-17 13-14 14-15 15-16 16-17
exact/norm bonds :
3-7 4-9 7-8 8-9
exact bonds :
8-10 10-11 11-12
normalized bonds :
1-2 1-6 2-3 3-4 4-5 5-6 12-13 12-17 13-14 14-15 15-16 16-17
isolated ring systems :
containing 1 : 12 :

G1:C,H

Match level :
1:Atom 2:Atom 3:Atom 4:Atom 5:Atom 6:Atom 7:Atom 8:Atom 9:Atom 10:CLASS 11:CLASS 12:Atom 13:Atom 14:Atom 15:Atom 16:Atom 17:Atom

L6 STRUCTURE UPLOADED

=> d l6
L6 HAS NO ANSWERS
L6 STR



G1 C,H

Structure attributes must be viewed using STN Express query preparation.

=> s l6 full
FULL SEARCH INITIATED 10:03:04 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED - 769 TO ITERATE

100.0% PROCESSED 769 ITERATIONS 163 ANSWERS
SEARCH TIME: 00.00.01

L7 163 SEA SSS FUL L6

=> file caplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	172.55	352.73

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-0.78

FILE 'CAPLUS' ENTERED AT 10:03:26 ON 30 APR 2007
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FILE COVERS 1907 - 30 Apr 2007 VOL 146 ISS 19
FILE LAST UPDATED: 29 Apr 2007 (20070429/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply.
They are available for your review at:

http://www.cas.org/infopolicy.html

=> s l7 full
L8 10 L7

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=> s l8 and py<2003
      22885160 PY<2003
L9            2 L8 AND PY<2003

=> d ibib abs hitstr tot
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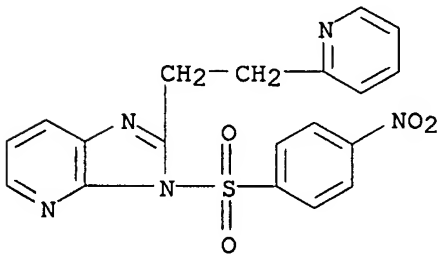
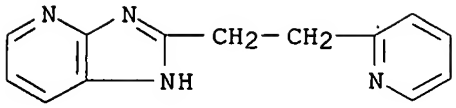
L9 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1999:284034 CAPLUS
DOCUMENT NUMBER: 131:82668
TITLE: Synthesis and antiproliferative activity of some N-sulfonated-2-substituted benzimidazoles and imidazo[4,5-b]pyridines
AUTHOR(S): Garuti, Laura; Varoli, Lucilla; Cermelli, Claudio; Baggio, Giosue; Lupo, Lucia; Malagoli, Monica; Castelli, Mario
CORPORATE SOURCE: Department of Pharmaceutical Science, University of Bologna, Bologna, I-40126, Italy
SOURCE: Anti-Cancer Drug Design (1998), 13(8), 969-980
CODEN: ACDDEA; ISSN: 0266-9536
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Some N-sulfonated-2-substituted benzimidazoles and imidazo[4,5-b]-pyridines were synthesized and tested in vitro for antiviral and antiproliferative activity. None of the compds. had antiviral properties. However, 3 of them inhibited the proliferation of leukemia and lymphoma cell lines at micromolar concns. The maximum potency of antiproliferative activity is correlated with the presence of an ethylenic spacer between the 2 heterocycles.

IT 229468-81-9P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(synthesis and antiproliferative activity of N-sulfonated-2-substituted benzimidazoles and imidazo[4,5-b]pyridines)
RN 229468-81-9 CAPLUS
CN 3H-Imidazo[4,5-b]pyridine, 3-[(4-nitrophenyl)sulfonyl]-2-[2-(2-pyridinyl)ethyl]- (9CI) (CA INDEX NAME)



IT 229468-73-9P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(synthesis and antiproliferative activity of N-sulfonated-2-substituted benzimidazoles and imidazo[4,5-b]pyridines)
RN 229468-73-9 CAPLUS
CN 1H-Imidazo[4,5-b]pyridine, 2-[2-(2-pyridinyl)ethyl]- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1997:549356 CAPLUS
DOCUMENT NUMBER: 127:152950
TITLE: Multiple unit effervescent dosage forms comprising proton pump inhibitor
INVENTOR(S): Lundberg, Per Johan
PATENT ASSIGNEE(S): Astra Aktiebolag, Swed.; Lundberg, Per Johan
SOURCE: PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9725030	A1	19970717	WO 1996-SE1738	19961220 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
SE 9600073	A	19970709	SE 1996-73	19960108 <--
SE 512835	C2	20000522		
CA 2214027	A1	19970717	CA 1996-2214027	19961220 <--
CA 2214027	C	20060905		
AU 9713242	A	19970801	AU 1997-13242	19961220 <--
AU 712325	B2	19991104		
BR 9607367	A	19971230	BR 1996-7367	19961220 <--
EP 814783	A1	19980107	EP 1996-944727	19961220 <--
EP 814783	B1	20031008		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
CN 1183716	A	19980603	CN 1996-193763	19961220 <--
HU 200000976	A2	20001128	HU 2000-976	19961220 <--
HU 200000976	A3	20001228		
IL 121653	A	20010808	IL 1996-121653	19961220 <--
AT 251451	T	20031015	AT 1996-944727	19961220
PT 814783	T	20040227	PT 1996-944727	19961220
ES 2208775	T3	20040616	ES 1996-944727	19961220
IN 1996DE02976	A	20050311	IN 1996-DE2976	19961227
ZA 9610939	A	19970708	ZA 1996-10939	19961230 <--
US 6132770	A	20001017	US 1997-793077	19970213 <--
NO 9704051	A	19971015	NO 1997-4051	19970903 <--
NO 319999	B1	20051010		

PRIORITY APPLN. INFO.: SE 1996-73 A 19960108
WO 1996-SE1738 W 19961220

OTHER SOURCE(S): MARPAT 127:152950

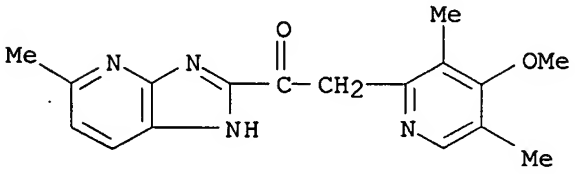
AB A new tabletted multiple unit effervescent dosage form containing an acid susceptible proton pump inhibitor in the form of the racemate, an alkaline salt thereof or one of its single enantiomers or an alkaline salt thereof, and

effervescent tablet constituents are claimed (Markush structure given). The proton pump inhibitor is preferably omeprazole or an alkaline salt thereof, or S-omeprazole or an alkaline salt thereof. Pellets comprising non-pareil cores 400, lansoprazole 400, hydroxypropyl Me cellulose 80, sodium lauryl sulfate 3, and water 1360 g were prepared The above pellets (100 g) were coated with a solution comprising hydroxypropyl Me cellulose 9, polyethylene glycol-6000 1, talc 18, 95% ethanol 250, and water 250 g. The above sub-coated pellets were enteric coated with a solution comprising hydroxypropyl Me cellulose phthalate 40, acetyltributyl citrate 8, cetanol 2, 95% ethanol 162, and acetone 378 g. The enteric-coated pellets were mixed with effervescent granules (preparation given) and compressed into tablets.

IT 193335-90-9
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(multiple unit effervescent dosage forms comprising proton pump inhibitor)

RN 193335-90-9 CAPLUS

CN Ethanone, 2-(4-methoxy-3,5-dimethyl-2-pyridinyl)-1-(5-methyl-1H-imidazo[4,5-b]pyridin-2-yl)- (9CI) (CA INDEX NAME)



=> d his

(FILE 'HOME' ENTERED AT 09:59:36 ON 30 APR 2007)

FILE 'REGISTRY' ENTERED AT 09:59:47 ON 30 APR 2007
L1 STRUCTURE UPLOADED
L2 5 S L1
L3 161 S L1 FULL

FILE 'CAPLUS' ENTERED AT 10:00:23 ON 30 APR 2007
L4 9 S L3 FULL
L5 1 S L4 AND PY<2003

FILE 'STNGUIDE' ENTERED AT 10:01:10 ON 30 APR 2007

FILE 'REGISTRY' ENTERED AT 10:02:32 ON 30 APR 2007
L6 STRUCTURE UPLOADED
L7 163 S L6 FULL

FILE 'CAPLUS' ENTERED AT 10:03:26 ON 30 APR 2007
L8 10 S L7 FULL
L9 2 S L8 AND PY<2003

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	13.02	365.75
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-1.56	-2.34

STN INTERNATIONAL LOGOFF AT 10:04:07 ON 30 APR 2007